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OBSERVATIONS ON THE MEAN PRESSURE AND THE CHARACTERS OF THE PULSE-WAVE IN THE CORONARY ARTERIES OF THE HEART.

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WHILE for a considerable number of years careful studies of the blood-flow in various arteries of the mammalian body have been made under different conditions, the arteries of the heart itself have remained in an exceptional position. The average pressure and the pulse characters in them have been unknown, in spite of the recognized fact that great interest and importance belong to their study.

The following pages give an account of experiments undertaken with the object of gaining some knowledge of these points, and contain, we believe, a description of the first successful attempt to record graphically, as in other arteries, the blood-pressure and its variations in the arteries of the heart. They were begun in the first place for the purpose of testing the theory of Thebesius—a theory independently propounded and warmly supported in recent times by Brücke, and others, concerning the physiology of the aortic semilunar valves. According to this theory, during ventricular systole the thin flaps of the valve are pressed upwards and cover the mouths of the coronary arteries, completely closing them, so that blood can enter these vessels only during the time of ventricular diastole and during that small portion of the systolic period which is occupied by the valve in travelling from its diastolic position across the mouth of the aorta, to its systolic position against the aortic wall and over the mouths of the coronaries. Observations on the spirting of blood from a cut coronary artery have shown this to be synchronous with systole of the ventricle ; but to the value of these observations Brücke[1] has raised two objections. First, that merely opening the pericardium is enough to destroy the normal action of the heart and consequently of the valve under consideration; and, second, even supposing it does not thus interfere, that the brief period

[1] *Vorlesungen*, 1881, S. 185.

before the valve-flap closes over the coronary (and during which the coronary blood must share the rise of aortic pressure due to commencing systole) is quite sufficient to cause a systolic spirting, especially if the outflow from the heart vessels is hindered by the increasing cardiac contraction compressing the smaller branches in the heart walls. The first objection seems to us trivial when only a small slit over the artery is made in the pericardial sac; but the second is more formidable. We know that in the first stage of the systole, before the valve can close over them, a rise of pressure must take place in the coronary trunks. To urge, therefore, that mere observation of spirting from the cut end of such a trunk can settle the question, is to claim that the unaided eye can distinguish between a rise of pressure only occurring early in the systolic period and a rise caused directly by the systole of the ventricle and lasting all through it. Considering how little time is needed for a complete contraction this is clearly impossible. Again, with our previous ignorance of the events transpiring in the cardiac vessels, and with no experimental evidence of increase or diminution of resistance in the smaller twigs during the systole, it is quite conceivable that some spirting might occur during that period, owing to the simultaneous effects of hindrance to the outflow and of increased pressure exerted upon the vessels by the contracting tissue. In a case like this, which calls for accurate observation and comparison, the graphical method is the only satisfactory one ; and as numerous attempts to settle the question at stake, based on other methods, have given rise to great diversity of opinion, we set to work to obtain, if possible, simultaneous records of the blood-pressure and pulse-waves in the coronary and carotid arteries.

Although the opposite view has from time to time been upheld by many anatomists and physiologists, nevertheless Brücke has so skilfully defended his theory that it is accepted by many physiologists to-day ; we, at least, prepared for our experiments with a decided leaning toward his view, and began work in the hope of establishing it more firmly. The results of our investigation, however, have forced us to believe that the semilunar valve does not act as Brücke supposes, and that his theory is no longer tenable. Apart, however, from this point, we venture to believe that the work possesses interest of its own ; and that the discovery that it is quite possible to get tracings of the blood-pressure in the arteries of the dog's heart, lays open a considerable field for investigations upon the mammalian heart in general—an organ which has hitherto been somewhat baffling to the physiologist.

Our experiments have all been made on dogs placed under the influence of a full, or rather an extreme, dose of morphia—from one to two grams of the acetate given subcutaneously in watery solution. While this drug greatly slows the respirations and somewhat later, to a certain extent, the rate of the heart's beat, it seems in no way to impair the vitality of this organ ; if anything it appears rather to increase its capacity for bearing insults—a matter deserving of further investigation. The animal having been put very completely under the influence of the drug, tracheotomy was performed, a cannula placed in one carotid artery, and the pneumogastric nerve of the same side exposed and divided so that its peripheral end was ready for stimulation.

An incision was then made in the middle line along the manubrium of the sternum ; the muscles, &c., were dissected from the first pair of costal cartilages, and (the apparatus for artificial respiration having been connected with the windpipe) the cartilages of the first pair of ribs and the bit of sternum between them were removed, thus laying bare the apex of the chest cavity, which was then opened. The artificial respiration was now stopped for a few seconds, so that the lungs might collapse and thus expose on each side the internal mammary artery, running along the exterior of the mediastinum and the remnant of the thymus, to the ventral aspect of the chest wall opposite the second costal cartilage. These arteries having been tied, the incision along the middle line was prolonged backwards and the skin and muscles reflected on each side so as to expose the rib cartilages. This operation is usually accompanied by only an inconsiderable venous oozing after the internal mammary arteries have been secured in the manner just mentioned.

The sternum and costal cartilages were then removed, care of course being taken not to injure the lungs. The next step was to stitch the pericardium to the chest wall in order to support the heart and prevent its receding too much when the lungs empty during expiration.

Branches of the coronary artery can now be seen through the pericardium, and a window is so cut in that membrane as to expose a branch which seems suitable, while all the rest of the heart remains protected and supported by its sac.

So far the operative procedures are tedious but present no special difficulty ; but to lay bare the coronary branch and to fix the cannula in it while the heart continues to beat is much more troublesome, since any carelessness in these operations is apt to so far injure the heart as to destroy its normal beat and throw the ventricles into incoordinate fibrillar contractions, from which we have never seen them recover. The success

of the attempt depends largely on the animal; in the most favourable cases the left coronary artery, after giving off its transverse branch, which runs along the auriculo-ventricular groove, passes along the septum ventriculorum on the ventral aspect of the heart, and gives off near the base of the ventricle a considerable branch to the right, which runs with a vein on each side of it, and is covered only by the visceral layer of the pericardium and some fat. Into this branch the cannula is inserted, and the blood carried by the main trunk and its remaining branches serves perfectly to keep the heart beating vigorously for several hours, as we have repeatedly found. In other cases the artery does not give off this one main branch, but (especially in large dogs) runs along the ventricles, giving off small twigs right and left which are too minute for the convenient introduction of a cannula, and are, moreover, often covered by a thin layer of the musculature of the heart in addition to the pericardium. This muscular layer adds greatly to the difficulty of successfully isolating the artery, for any wound to the proper cardiac substance about the vessels seems more fatal to the organ than anything else. Soon after such an injury it almost invariably exhibits periodic beats for a short time and then the ventricle passes into a state of fibrillar contraction. The well-known fact that needles may be thrust into many parts of the heart without essentially influencing its beat for a long time, inclines us to the belief that the result in the cases to which we refer is perhaps due to the injury of nerve trunks which may run in the heart near its arteries and which are torn with the muscle, rather than to direct injury of the muscular substance ; but we have not yet had an opportunity to examine this point.

A suitable coronary branch having been found, the next step is the most difficult in the operation, viz., to tear through the visceral pericardium over the artery without opening that vessel or its accompanying veins ; for the membrane is so smooth and tightly stretched that it is not easy to catch hold of ; and then so tough that it is difficult to penetrate. Our method is as follows:—All being ready, the pneumogastric trunk is stimulated so as to stop the heart's beat, and the artificial respiration simultaneously suspended so as to avoid movements of the heart due to contractions and expansions of the lungs. With a sharp-pointed pair of forceps the pericardium over the artery is seized and a hole torn through it by means of a needle ; once this aperture is made through the tough membrane without injuring any of the vessels the rest of the operation is comparatively easy. The stimulation of the pneumogastric is stopped and the artificial respiration resumed for a moment or two ; then the heart-

beat and breathing are again suspended, the edge of the hole is taken in the forceps and the membrane over the artery slit up toward the base of the heart by a very fine-bladed knife. From time to time, as the heart begins to beat in spite of stimulation of the pneumogastric, the nerve is allowed to rest and the respiration is resumed, and in this way the alternate stimulation and rest are repeated as often as may be necessary in order to expose a sufficient length of the artery, to place ligatures around it, and insert a cannula in the manner adopted for any other artery. The carotid is then connected with one mercury manometer, the coronary branch with another, and, the pens being arranged so as to write exactly over one another, tracings are taken on the kymographion.

The mode of connection of the arteries with the manometers demands a word. In the first place, the three inches of the arterial end of the connecting tube between the coronary and its manometer consist of highly flexible rubber tubing. This, no doubt, slightly modifies the pulse-waves on the tracing, but it gives to the heart free play during each beat, since the flexible tube offers no restraint but yields readily. This soft tubing is succeeded by a glass tube, which is firmly held by a solid support, so that no locomotion of the tubing occurs beyond this point.

Movement of the bit of flexible tubing attached to the cannula does slightly alter the level of mercury in the manometer, but, as we have satisfied ourselves by careful examination, causes no features in the tracing which can be mistaken for a pulse-wave. Beyond the piece of glass tubing mentioned above, the connecting arrangement is similar for the two arteries.

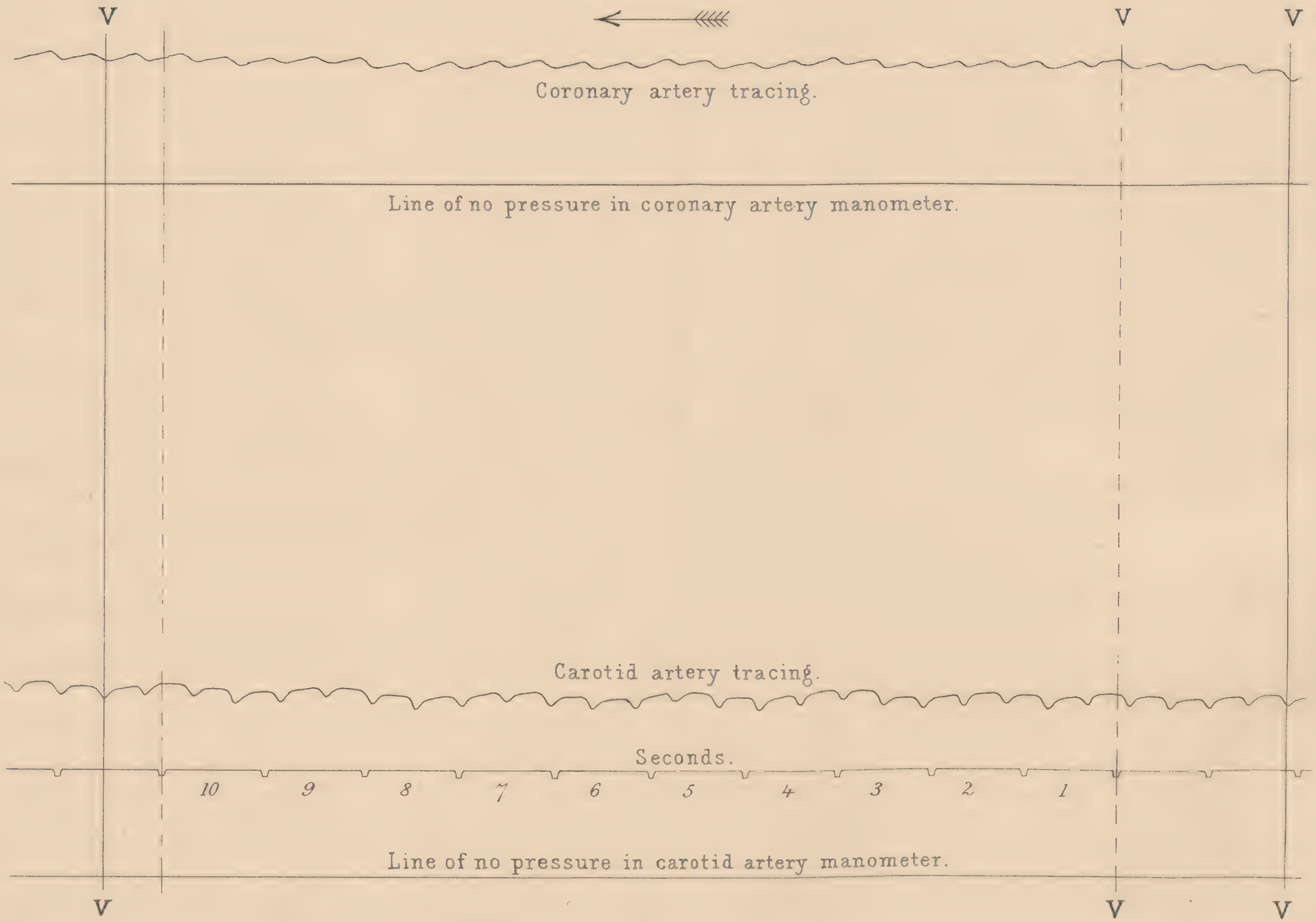
To get a true base-line, or line of no pressure, for each manometer gave us some little trouble. The base-line is often taken as that drawn by the pen when the mercury stands at the same height in both legs of the manometer, but this is seldom correct. If the end of the connecting apparatus attached to the artery be above the level of the mercury in the limbs of the manometer with which it is joined, the weight of the liquid in it will affect that level, making it sink in the nearer and, of course, rise in the farther limb which bears the pen. If, on the other hand, as is more often the case, the arterial end of the connecting tube be below the level of the mercury in the gauge the tube acts like a siphon-tube; the mercury rises somewhat in the proximal limb, and sinks to the same extent in that which carries the pen, so that in either case the base-line drawn with the two mercury columns level will be incorrect.

As we wished especially to compare the amount of arterial pressure

in the coronary with that in the carotid, we had to eliminate such errors, and the more so because the manometer attached to the coronary artery was invariably above the one connected with the carotid, and so the siphon action (for the ends of the tubes farthest from the kymographion were always below the levels of the mercury in the manometers) was considerably greater. The method which we adopted gives, we think, absolutely true results. Having finished an experiment, we stopped the artificial respiration, and let the animal die of asphyxia, the manometers being meanwhile shut off from connection with the arterial system. When the animal was quite dead, and all traces of arterial pressure had disappeared, the communication with the manometers was again opened, and the pens, of course, fell with the mercury to the level which corresponded to zero arterial pressure : we, of course, satisfied ourselves that there were no clots in the apparatus. The pens were then turned away from the paper, which was next recoiled on the drum until the beginning of the record of the experiment was reached ; then, the pens being turned back again, the kymographion was started once more and each pen drew its own base line, being still connected with its artery and the position of the animal being the same as during the experiment. It has been suggested to us that the base line so obtained may not be reliable as some arterial pressure might still remain in either the carotid or coronary vessel, or in both, after general death ; but this objection we think will not bear examination. After death from asphyxia, as is well known, the arterial system, at least in its larger trunks, is extremely empty ; a few minutes after its occurrence one may cut the aorta without the slightest spirt of blood resulting, and, indeed, even almost without bleeding at all ; and the carotids, subclavians, and other large arterial trunks are obviously collapsed and empty. That under such circumstances there should be any arterial pressure possibly remaining in arteries in free and direct connection with the aorta is not conceivable.

A description of the tracings taken on the kymographion (Figs. 1—5, Pl. VIII., IX., X.) will serve best to show our results. The tracings, in fact, speak for themselves, and have been selected from a considerable number which all perfectly agree with them as to the conclusions to which they lead; we have never obtained a single contradictory record. The pulse synchronism and the similarity of the pulse-waves in the carotid and coronary under different amounts of blood-pressure and with various rates of heart-beat is remarkable throughout. In Fig. 1, Pl. VIII., we have a pulse rate of 132 per minute, and complete synchronism in the two arteries; the mean pressure in the former being 62 mm. of Hg.

Fig. 1.



F. Huth, Lith[r] Edin[r]

Fig. 2.

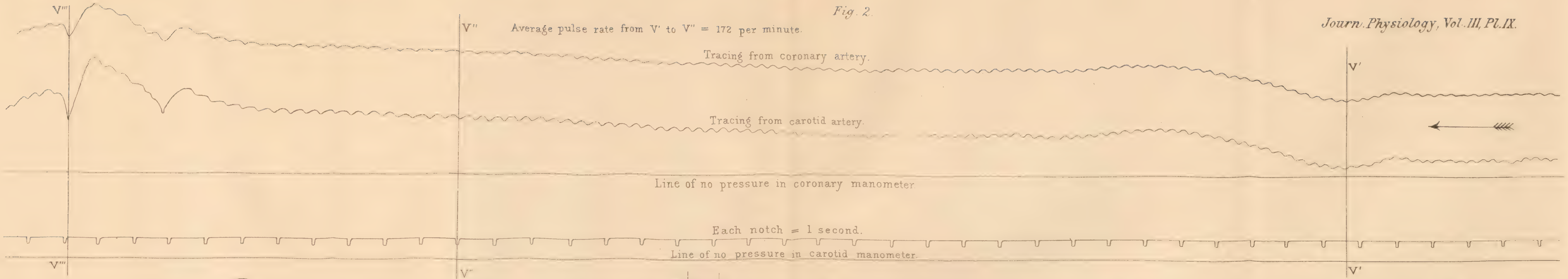

V'''
V''
Average pulse rate from V' to V'' = 172 per minute.
Tracing from coronary artery.
V'
Tracing from carotid artery.
Line of no pressure in coronary manometer.
Each notch = 1 second.
Line of no pressure in carotid manometer.
V'''
V''
V'

Fig. 3.

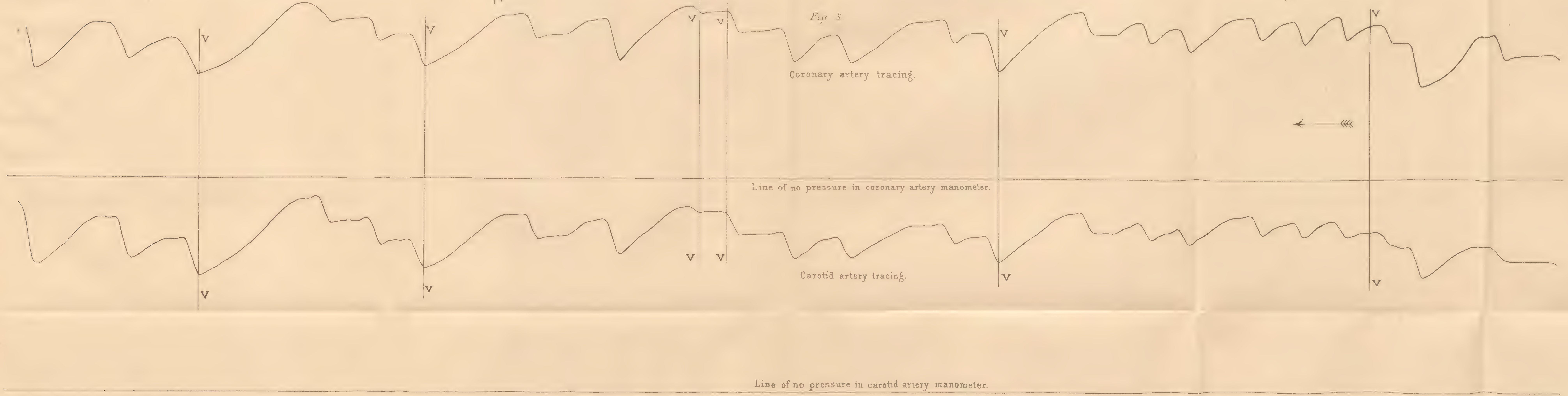

V
V
V
V
V
V
Coronary artery tracing.
Line of no pressure in coronary artery manometer.
V
V
Carotid artery tracing.
V
V
V
V
Line of no pressure in carotid artery manometer.

Fig. 4.

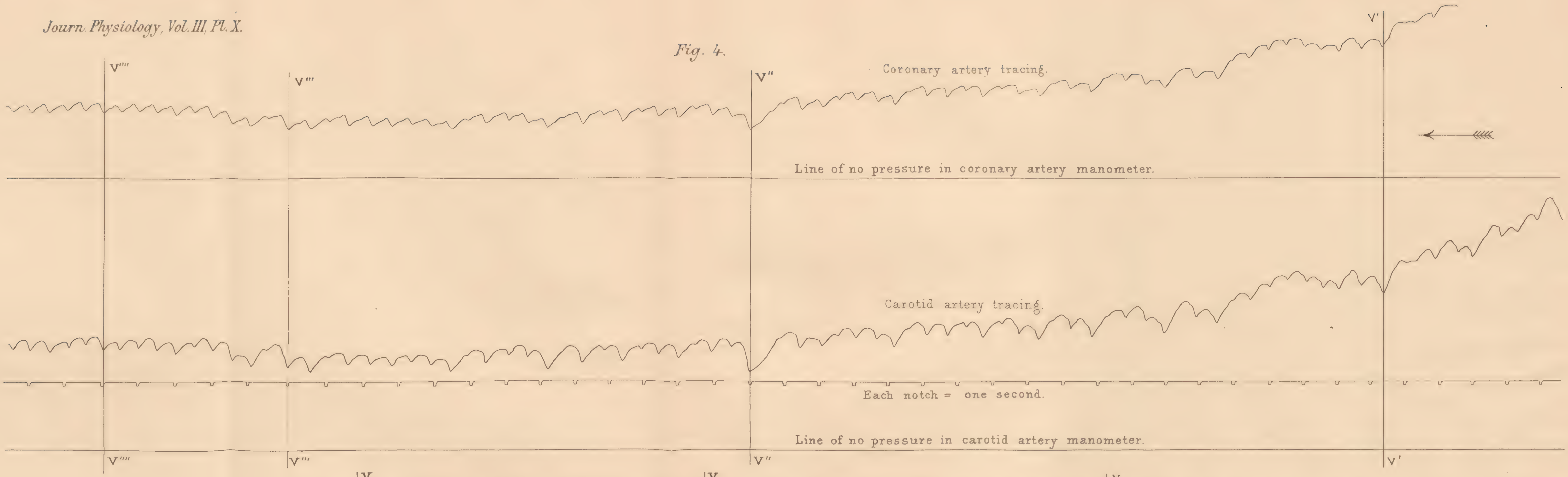
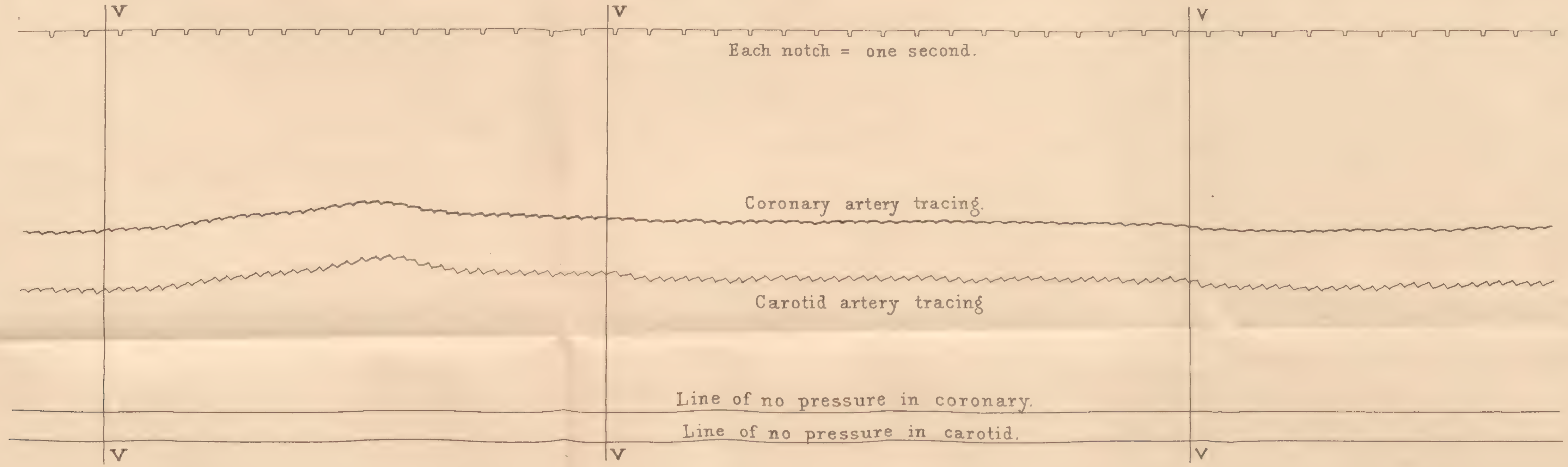

V''''
V'''
V''
V'
Coronary artery tracing.
Line of no pressure in coronary artery manometer.
Carotid artery tracing.
Each notch = one second.
Line of no pressure in carotid artery manometer.
V''''
V'''
V''
V'

Fig. 5.


V
V
V
Each notch = one second.
Coronary artery tracing.
Carotid artery tracing
Line of no pressure in coronary.
Line of no pressure in carotid.
V
V
V

and in the latter 42. The verticals, vv, cut all the tracings at points corresponding to the same moment of time. In Fig. 2, Pl. IX., is a tracing taken with a quicker pulse, about 172 per minute. At v′, artificial respiration was stopped so as to get a dyspnœic rise of arterial pressure. As the verticals show, this does not disturb in the least the synchronism or similarity of the pulse-waves in the two arteries. Mean pressure in coronary, 46 mm. of Hg., and in carotid 56, at the beginning, rising to 100 mm. and 120 mm. respectively just before v‴.

Fig. 3, Pl. IX., gives simultaneous tracings from the two arteries during extreme dyspnœa, with greatly slowed pulse and very high blood pressure, rising in the part of the tracing given to 120 mm. of Hg. in the coronary artery and to 132 in the carotid. Ultimately the pressure rose still higher, and drove the pen attached to the coronary vessel off the top of the paper, so that a record could not be obtained. The accuracy with which each tracing reproduced the other during all the variations of pressure and pulse-rate which occurred during this observation is very remarkable, and seems to make it certain that the pressure in each artery is directly determined by the same cause, viz., aortic pressure. The contracting ventricle might conceivably increase pressure in the coronary vessels by compressing them; but variations thus produced cannot be imagined as agreeing so perfectly with the variations in carotid pressure (which, on such a theory, must be under different influences) as do those given in this figure.

Unfortunately a seconds' pen was not connected with the kymographion on this occasion, so that the pulse-rate cannot be stated accurately; but by taking an average from the rate of movement in other cases it may be set down as about 60 without any great error.

In Fig. 4, Pl. X., is given a tracing taken soon after the resumption of artificial respiration, which had been interrupted long enough to produce (as seen to the right of the tracing) a considerable dyspnœic rise of arterial pressure. Well marked and similar Traube's curves are seen on each tracing, and also the synchronous pulse in both arteries. This synchronism is maintained throughout all changes of cardiac rhythm and blood pressure.

In Fig. 5, Pl. X., is a tracing in which the coronary pressure is higher than the carotid (76 mm. against 64 mm. Hg.). This may perhaps be due to our having taken in this case a coronary branch nearer the main stem than usual; but it may also, and more probably, be vasomotor. The heart arteries have a very active system of these nerves, as any one who experiments with them will soon observe. Not unfre-

quently on laying bare a coronary branch that seemed suitable for inserting the cannula we have found it apparently so small that our endeavour seemed hopeless ; and then in a minute or two it would dilate again to at least double its previous diameter. If it be borne in mind that the coronary branch used was always but a small twig of the whole coronary system, it seems possible that great constriction in the rest of the branches might so oppose the blood flow as to raise the pressure almost up to that in the aortic arch, and so bring it above that in the carotid[1].

In other respects the tracing illustrates the same points as those reproduced in the preceding figures. The heart was beating 148 per minute.

We find then that whether the heart beats slow or fast, and whether arterial pressure be high or low, every feature of the carotid pulse is simultaneously given in the coronary. No doubt, with a faster-travelling roll of paper the synchronism would not be perfect, as the carotid vessel is farther from the heart, but the pulse-wave travels so fast that this could not be expected to be shown on the kymograph.

There is, however, no trace of any alternation in the pulse-waves, such as would seem necessarily to follow from an occlusion of the mouths of the coronary arteries during the ventricular systole, and such as the kymograph would certainly show if it existed.

The argument which was used effectively against conclusions drawn from observations upon spirting coronary arteries, may be brought perhaps to bear upon our work, viz. : that in the earliest stage of contraction of the ventricle, the coronary shares with the carotid the general rise of pressure in the arterial system, because the valve has not yet closed over its mouth ; and that, in consequence, it is to be expected that the two pens which have travelled during the diastole of the previous undulation together shall together begin their systolic journey on the new pulse-wave. This is, no doubt, quite true, and we have no objection to the argument as far as it goes. It leaves off, however, where our work begins, and does not affect the real point of the question, though it emphasizes the necessity for exact tracings which can be studied leisurely.

[1] We have recently endeavoured to discover the source of the vaso-constrictor nerves of the heart, by connecting cannulæ with carotid and coronary arteries and then observing if a relative rise of coronary pressure could be brought about by stimulating extrinsic cardiac nerves. So far our experiments have been confined to the accelerators and have been entirely negative. We got the acceleration of the pulse rate, but no rise or fall in coronary pressure, which was not exactly duplicated on the tracing from the carotid manometer.

Since the coronary artery is freely exposed to aortic pressure during all of the diastole and during the first fraction of the systole of the ventricle, we are not surprised to find, at that time, on the tracings complete agreement between carotid and coronary pulses ; they are caused by the same things and are therefore similar. If now we turn to the tracings described during the major portion of the systolic period, and find them duplicates one of the other, alike in form and synchronous in characters, it is hard to believe that they also are not directly dependent on the same immediate cause, *i.e.*, aortic pressure. For if the valve closes as Brücke believes, the forces acting upon the two arterial contents are no longer identical ; the carotid is still marking an increasing pressure due to the outflow of blood from the energetically contracting ventricle ; but the coronary, cut off by the valve from influx of blood, is put under other conditions. It is not supposable that the ventricle acting upon the carotid directly through the aorta should cause it to trace a pressure curve precisely like one drawn at the same time by the coronary, upon which it is acting only indirectly (*i.e.*, by raising intraventricular pressure, and so causing extra compression of the vessels in the heart substance). Nor is it conceivable that the coronary artery should have its mouth suddenly closed at one instant during the period of rising pulse-wave, and still go on tracing undisturbed an uniform rise of pressure. Under such circumstances some deformation of the coronary curve, some irregularity in the tracing, must take place. Again, after the systole is over and the valves rebound to their position over the mouth of the aorta, a moment would come (when the period of highest carotid pressure was just past) when the coronary artery would suddenly be opened and blood would be driven into it.

An injection of blood into the previously closed coronary system at this moment ought surely (even if it did not, as may be urged, raise arterial pressure in the coronary artery, because the cardiac muscle was relaxing and making the coronary circuit easier of passage) to show itself in some break or rise, or other special feature in the pressure-changes at that moment occurring in the vessel ; the tracing from the coronary vessel (now for the first time receiving blood) could not exactly agree in every respect with the tracing from the carotid artery, which is simultaneously emptying itself steadily and regularly under the force of arterial elasticity. We find, however, nowhere any indication of such events ; the coronary tracing is always a duplicate of the carotid under all circumstances, and there is no sign of any periods when great circulatory changes (such as are involved in the supposition that the

mouths of the coronary vessels are alternately closed and opened) are taking place in the coronary artery.

We are therefore forced to conclude that those are in the right who have maintained that the flaps of the semilunar valve are never pressed completely back against the aortic wall during systole of the ventricle. Finally we may point out that the tracings show the pressure-changes in the coronary system to be very much like those in any other branch of the aortic system—the carotid for example. It may be added in conclusion that though forced to differ from Brücke in regard to any interference of the semilunar valve with the circulation in the coronary system, our observations in no way contradict his teaching that during ventricular diastole blood flowing into the coronary arteries aids in distending the flaccid heart. This is probably true. The complete "*Selbststeuerung*" is however no longer tenable; the arteries of the heart are not emptied during the ventricular diastole so as to diminish the resistance to contraction but are at that time tensely filled. Moreover, as our tracings show, the little increment of pressure during the systole of a single beat when compared with the entire mean pressure constantly at work in the coronary system is so small that after all not much would be gained by blocking the mouths of the arteries in order to avoid it.